08/765026
Search results for Paper #38

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Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Term: L4 and gene near therap$ and (diabetes or hypertension or parkinson or retinopathy or huntington)

Display: 100 **Documents in Display Format:** - **Starting with Number** 1

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Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L6	L4 and gene near therap$ and (diabetes or hypertension or parkinson or retinopathy or huntington)	19	L6
L5	L4 and gene near therap$	39	L5
L4	(superoxide near dismutase$ or SOD or "SOD-1" or CuZn near SOD or CuZnSOD) near10 treat$ and adenovir$	57	L4
L3	(superoxide near dismutase$ or SOD or "SOD-1" or CuZn near SOD or CuZnSOD) and adenovir$	1045	L3
L2	(superoxide near dismutase$ or SOD or "SOD-1" or CuZn near SOD or CuZnSOD) near20 adenovir$	23	L2
L1	(superoxide near dismutase$ or SOD) near10 adenovir$	18	L1

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Search Results - Record(s) 1 through 39 of 39 returned.

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Terms	Documents
L4 and gene near therap$	39

Previous Page Next Page

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set hi ;set hi
HILIGHT set on as ''
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? begin 5,6,55,154,155,156,312,399,biotech,biosci
>>>         135 is unauthorized
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      Set  Items  Description
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? s (superoxide (n) dismutase or SOD or "SOD-1" or CuZnSOD or CuZn (n) SOD) (5n)
adenovir?
Processed  30 of  34 files ...
Completed processing all files
          331148  SUPEROXIDE
          206302  DISMUTASE
          204410  SUPEROXIDE(N)DISMUTASE
           84895  SOD
             166  SOD-1
            2838  CUZNSOD
            6156  CUZN
           84895  SOD
            2439  CUZN(N)SOD
          193750  ADENOVIR?
      S1     439  (SUPEROXIDE (N) DISMUTASE OR SOD OR "SOD-1" OR CUZNSOD OR
                  CUZN (N) SOD) (5N) ADENOVIR?
? s s1 not py>1995
Processing
Processed  10 of  34 files ...
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>>>  or undefined in one or more files.
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Processing
Completed processing all files
             439  S1
        46642802  PY>1995
      S2      13  S1 NOT PY>1995
? d s2/9/1-13
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DIALOG(R)File   5:Biosis Previews(R)
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04748390   BIOSIS NO.: 000080051517
MODIFICATION OF DOPA TOXICITY IN HUMAN TUMOR CELLS
AUTHOR: PARSONS P G
AUTHOR ADDRESS: QUEENSLAND INSTITUTE OF MEDICAL RESEARCH, HERSTON,
  QUEENSLAND, AUSTRALIA 4006.
JOURNAL: BIOCHEM PHARMACOL 34 (10). 1985. 1801-1808. 1985
FULL JOURNAL NAME: Biochemical Pharmacology
CODEN: BCPCA
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: A variety of factors were found to modify the toxicity of L-dopa
  in HeLa cells (D37 16 .mu.M) and in dopa-sensitive, nonpigmented human
  melanoma cells (MM96) (D37 5 .mu.M) having a similar size and doubling
  time. Dopa toxicity was decreased by concurrent treatment with superoxide
  dismutase, peroxidase or catalase, by erythrocytes, or by hypoxia.

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      Display 2/9/1     (Item 1 from file: 5)
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  Toxicity could be increased by the enzyme inhibitors L- and
  D-penicillamine, sodium diethyldithiocarbamate or 3-amino-1,2,4-triazole.
  The 2 cell lines had similar levels of superoxide dismutase and
  peroxidase; in 6 human melanoma lines, no correlation was found between
  dopa killing and tyrosinase activity as determined either by formation of
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dopa from tyrosine or by formation of melanin from dopa. Uptake of L-dopa was similar in HeLa and MM96 cells, and the toxicity of D-dopa was the same in both lines as that of the L-isomer. Dopa decomposed within 12 h in culture medium, the rate and products being influenced by addition of the above enzymes and by the cell density. Dopa-melanin and medium containing decomposed dopa were also selectively toxic to MM96 cells. Adenovirus 5 was used in 2 different ways to assess the relative importance of DNA damage and inhibition of DNA synthesis by dopa. Viral replication was found to be unaffected in cells being treated with dopa but was strongly inhibited in cells treated with the DNA polymerase inhibitor cytosine arabinoside. Secondly, the virus was itself inactivated by treatment with dopa for 24 h (D37 1.3 mM); similar dose

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DIALOG(R)File 5:Biosis Previews(R)

response curves were obtained for replication of dopa-treated virus in untreated HeLa or MM96 cells. These results show that the initial events of dopa toxicity occur outside the cell and lead to the formation of a stable, toxic product (probably melanin) which does not strongly inhibit DNA polymerase activity. Melanoma hypersensitivity was not due to differences in oxygen-metabolizing enzymes, dopa uptake, or DNA repair.

DESCRIPTORS: MELANOMA CELLS HELA CELLS L PENICILLAMINE D PENICILLAMINE SODIUM-DIETHYLDITHIOCARBAMATE 3 AMINO-1 2 4-TRIAZOLE **SUPEROXIDE DISMUTASE** PEROXIDASE **ADENOVIRUS** 5 DNA DAMAGE
CONCEPT CODES:
02508 Cytology and Cytochemistry-Human
10808 Enzymes-Physiological Studies
13012 Metabolism-Proteins, Peptides and Amino Acids
22005 Pharmacology-Clinical Pharmacology (1972-)
22504 Toxicology-Pharmacological Toxicology (1972-)
24008 Neoplasms and Neoplastic Agents-Therapeutic Agents; Therapy

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DIALOG(R)File 5:Biosis Previews(R)

10010 Comparative Biochemistry, General
10012 Biochemistry-Gases (1970-)
10060 Biochemical Studies-General
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
11108 Anatomy and Histology, General and Comparative-Microscopic and Ultramicroscopic Anatomy
13014 Metabolism-Nucleic Acids, Purines and Pyrimidines
15004 Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies
22003 Pharmacology-Drug Metabolism; Metabolic Stimulators
33506 Virology-Animal Host Viruses
BIOSYSTEMATIC CODES:
02210 Adenoviridae (1979-)
86215 Hominidae
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA):
Microorganisms
Viruses

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  Animals
  Chordates
  Vertebrates
  Mammals
  Primates
  Humans

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Display 2/9/2 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

00976947 Genuine Article#: FK380 Number of References: 36
Title: THE E1B ONCOGENE OF ADENOVIRUS CONFERS CELLULAR-RESISTANCE TO CYTOTOXICITY OF TUMOR-NECROSIS-FACTOR AND MONOCLONAL ANTI-FAS ANTIBODY
Author(s): HASHIMOTO S; ISHII A; YONEHARA S
Corporate Source: MEIJI INST HLTH SCI,540 NARUDA/ODAWARA 250//JAPAN/; TOKYO METROPOLITAN INST MED SCI,DEPT CELL BIOL,BUNKYO KU/TOKYO 113//JAPAN/
Journal: INTERNATIONAL IMMUNOLOGY, 1991, V3, N4, P343-351
Language: ENGLISH Document Type: ARTICLE
Geographic Location: JAPAN
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: IMMUNOLOGY
Abstract: The cell lines KB8, 16, and 18 are KB cells which constitutively express adenovirus type 2 (Ad2) E1a, E1a plus E1b, and E1b genes, respectively. We show here that KB18 cells are completely resistant to cytolysis by tumor necrosis factor (TNF) or anti-Fas, although KB8 and KB or KB16 are highly and moderately sensitive, respectively. The

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levels of receptors for TNF and anti-Fas of KB18 were almost the same as compared with those of KB or other KB-cell lines. Expression of manganous superoxide dismutase (MnSOD) mRNA in KB18 was about 20-fold higher than that in KB or KB8 cells. KB, HT29, and A673 cells infected with dl337 (an Ad5 mutant defective in E1b function) are highly sensitive to TNF or anti-Fas, although wild-type Ad2-infected cells are resistant. Our results indicate that the E1b oncogene can confer cellular resistance to cytolysis by either TNF or anti-Fas in both KB cells and adenovirus-infected human cell lines through influencing intracellular events including regulation of MnSOD genes. Furthermore, we describe how anti-Fas mimics only the cytolytic activity of TNF, whereas TNF also has many other biological activities.
Descriptors--Author Keywords: **ADENOVIRUS** E1A GENE; MANGANOUS **SUPEROXIDE DISMUTASE**; FAS-ANTIGEN; KB CELLS
Identifiers--KeyWords Plus: MANGANOUS SUPEROXIDE-DISMUTASE; CYTO-TOXICITY; FACTOR TNF; BINDING; RECEPTORS; CELLS; HOST; GENE; DEGRADATION; EXPRESSION

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Research Fronts: 89-0312 003 (TUMOR NECROSIS FACTOR; ANTIVIRAL ACTIVITY; EFFECTS OF CYTOKINES)
  89-7486 001 (TUMOR NECROSIS FACTOR; LANGERHANS CELLS IN VACCINIA VIRUS-INFECTION; INTERFERON-GAMMA BINDING)

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- end of record -

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Display 2/9/3 (Item 1 from file: 266)
DIALOG(R)File 266:FEDRIP

00351692
IDENTIFYING NO.: 3P01NS37520-03S1 9002 AGENCY CODE: CRISP
Core--Vector
PRINCIPAL INVESTIGATOR: SAPOLSKY, ROBERT M
ADDRESS: STANFORD UNIVERSITY MED CTR R 161 STANFORD, CA 94305-5327
PERFORMING ORG.: STANFORD UNIVERSITY, STANFORD, CALIFORNIA
SPONSORING ORG.: NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE

FY : 2001 TYPE OF AWARD: Supplement (Type 3)
SUMMARY: The Vector core will produce herpes simplex virus (HSV)-based amplicon vectors, adenoviral vectors, and retroviral vectors. These will be used in Project 1 in m ixed neuronal/glial cultures and pure astrocyte cultures, as well as in Project 2, for in vivo ischemia studies. HSV and adenoviral vectors will be generated ex pressing the genes for CuZn-superoxide dismutase (SOD1), glutathione peroxidase (the prior two either individually, or in combination, Bcl-2, and Hsp70. All of the HSV and adenoviral vectors will be "bicistronic", expressing both the gene in

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question, as well as a reporter gene. For each experimental vector, the cogna te control vector will contain the same gene in question with a stop codon inser ted in its center, insuring non-expression. Similar controls will be generated f or the adenoviral vectors. Retroviral vectors to express SOD1, Mn-SOD (SOD2), Bc l-2 and Hsp70 will be produced. Control vector will express either the reporter gene beta-galactosidase or a stop codon version of the gene of interest. The ret roviral vectors for HSP70 and Bcl-2 have been constructed and they will be produ ced in the vector core. In addition new vectors to express SOD1 and SOD2 (separa tely) will be constructed and then produced. The purpose of the Vector Core will be to ensure a constant supply of vectors for the various groups, to insure qua lity control in the production of such vectors, and to troubleshoot problems, as they arise, in individual laboratories in the use of vectors.
DESCRIPTORS: biomedical facility; **superoxide dismutase**; stress protein; **Adenoviridae**; Alphaherpesvirinae; Retroviridae; transfection /expression vector; BCL2 gene /protein

- end of record -

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00351689
IDENTIFYING NO.: 3P01NS37520-03S1 0002 AGENCY CODE: CRISP
In vivo injury paradigms
PRINCIPAL INVESTIGATOR: STEINBERG, GARY K
ADDRESS: STANFORD UNIVERSITY MED CTR R 161 STANFORD, CA 94305-5327
PERFORMING ORG.: STANFORD UNIVERSITY, STANFORD, CALIFORNIA
SPONSORING ORG.: NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE
FY : 2001 TYPE OF AWARD: Supplement (Type 3)
SUMMARY: Recent progress in the area of stroke research suggests that a number of molecul ar mechanisms are intimately involved in the evolution of ischemic brain injury. Gene induction has been observed following ischemia, but the exact roles of man y are not yet well known. Some gene products are known to be detrimental to the cell while others are felt to be neuroprotective. The goals of this project are to define more precisely the roles of three classes of genes which may play neur oprotective roles. They are: the proto oncogene, bcl-2, antioxidant genes (sod-1 and gspx) and the

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stress protein, hsp70. In Project 2, we will utilize genetica lly normal animals and study 3 different in vivo models of ischemia (2 focal and one global models of cerebral ischemia). We will alter gene expression via gene

transfer using defective herpes simplex and adenoviral vectors. We will study t he limits and conditions under which gene over-expression may improve neuron sur vival. We hypothesize that injury due to some, but not other kinds of insults wi ll be attenuated with gene product over-expression, and that these observations will offer insight into the pathophysiology of cell death. We will examine wheth er gene transfer after the onset of injury is neuroprotective, and whether over- expression of Bcl-2 and antioxidant genes are protective against permanent as we ll as transient focal cerebral ischemia. We will also examine mechanisms underly ing neuroprotection, or lack of neuroprotection, by examining the participation of other gene products such as the stress proteins, caspases and Bcl-2 family pr oteins in response to gene over-expression and cerebral injury. We will also stu dy whether gene over-expression alters generation of superoxide and apoptosis. T hese novel approaches will hopefully add

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insight into the complex molecular proc esses involved in cerebral ischemia and may lead to the development of treatment s for stroke and other degenerative disorders.
DESCRIPTORS: laboratory rat; cell death; cerebral ischemia /hypoxia; transfection; protooncogene; gene induction /repression; immunocytochemistry; disease /disorder model; electron microscopy; antioxidant; **superoxide dismutase**; stress protein; **Adenoviridae**; Alphaherpesvirinae; transfection /expression vector; transient ischemic attack; neuroprotectant; neuropathology; BCL2 gene /protein

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DIALOG(R)File 266:FEDRIP

00351688
IDENTIFYING NO.: 3P01NS37520-03S1 0001 AGENCY CODE: CRISP
In vitro injury paradigms
PRINCIPAL INVESTIGATOR: SAPOLSKY, ROBERT M
ADDRESS: STANFORD UNIVERSITY MED CTR R 161 STANFORD, CA 94305-5327
PERFORMING ORG.: STANFORD UNIVERSITY, STANFORD, CALIFORNIA
SPONSORING ORG.: NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE
FY : 2001 TYPE OF AWARD: Supplement (Type 3)
SUMMARY: The protective potential of three groups of genes will be studied in ischemia-li ke injury of brain injury of brain cells from cortex, hippocampus, and striatum, in primary culture. First an antioxidant strategy will be tested by over-expres sing CuZn **superoxide dismutase** (SOD1) using herpes virus and **adenoviral** vectors to achieve rapid expression in neurons and astrocytes, and retroviral vectors fo r prolonged, stable expression in astrocytes. Whether acute expression can provi de protection will be tested. If this is not protective, the

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effect of prolonged stable expression and the use of bicistronic vectors, to rapidly express both S OD and a downstream antioxidant vectors, to rapidly express both SOD and a downs tream antioxidant enzyme, will be tested. We will test for protective effects, a s well as for induction of

other antioxidant enzymes. Whether protection correla tes with induction of other antioxidant enzymes will be determined. Under condit ions where protections seen the extent of oxygen radical production, lipid perox idation and changes in level of glutathione will be determined. Whether this gen e protects against necrotic or apoptotic forms of cell death will be determined. Whether this gene protects against necrotic or apoptotic forms of cell death wi ll be determined, as well as the time window in which expression can still prote ct, since it is important to develop therapeutic strategies that are effective a fter insults. Second, we will study the ability of Bcl-2 expression to protect i n the same injury paradigms. Oxidative status and the time window in which Bcl-2 can protect will be determined. Third we will study the ability of the inducibl e heat shock protein 70 (HSP70) to protect from these injury again analyzing the

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time window during which this gene can protect and whether it blocks apoptotic or necrotic cell death. Primary cultures are particularly useful for analyzing m echanisms of ischemic brain injury and mechanisms of protection at the cellular level. Primary cultures of neurons and glial cells and pure astrocyte cultures w ill be mad3e from hippocampus and striatum. Results will be compared with parall el studies carried out on primary cultures from neocortex. In addition, astrocyt e cultures will be produced from transgenic mice made with different gene dosage s of SOD1 or MnSOD (SOD2), including knockouts lacking these genes, to determine the importance of these enzymes for astrocyte survival of ischemia-like insults . These studies will provide fresh insight into possible mechanisms of protectio n by three candidate genes for anti- ischemic gene therapy tested in three brain regions.
DESCRIPTORS: laboratory mouse; transgenic animal; hippocampus; occipital lobe /cortex; brain cell; cell death; cerebral ischemia /hypoxia; enzyme induction /repression; gene therapy; disease /disorder model; glia; astrocyte; antioxidant; **superoxide dismutase**; stress protein;

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tissue /cell culture; **Adenoviridae**; Alphaherpesvirinae; transfection /expression vector; neuroprotectant; BCL2 gene /protein

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00341002
IDENTIFYING NO.: 5R01HL60132-03 AGENCY CODE: CRISP
LUNG RADIATION PROTECTION BY MNSOD--TRANSGENE THERAPY
PRINCIPAL INVESTIGATOR: GREENBERGER, JOEL S
ADDRESS: UNIV OF PITTSBURGH CANCER INST 200 LOTHROP STREET PITTSBURGH, PA 15213
PERFORMING ORG.: UNIVERSITY OF PITTSBURGH AT PITTSBURGH, PITTSBURGH, PENNSYLVANIA
SPONSORING ORG.: NATIONAL HEART, LUNG, AND BLOOD INSTITUTE
FY : 2001 TYPE OF AWARD: Noncompeting Continuation (Type 5)
SUMMARY: The lung is a major dose-limiting tissue in radiation therapy. We have demonstrated in mice that intratracheal infection of plasmid

liposomes or second generation replication-defective **adenovirus** constructs carrying the human manganese **superoxide dismutase** (MnSOD) transgene results in a significant decrease in both acute and chronic (alveolitis/fibrosis) damage by whole long irradiation. Both

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delivery systems demonstrated increased MnSOD mRNA levels in the airway prior to irradiation and decreased levels of transcripts for inflammatory cytokines that irradiation. We will now confirm the observations, and optimize in rigorous preclinical studies the efficiency and safety of pulmonary MnSOD transgene therapy using plasmid/liposomes for lung irradiation protection. Three specific are designed for proof of the principal that MnSOD transgene therapy will protect normal lung from irradiation damage. In the first specific aim C57BL/6J mice, which are heterozygous deletion recombinant-negative for murine MNSOD, will be compared will be compared with normal littermates for irradiation-induced organizing alveolitis. Our preliminary data show increased susceptibility of MnSOD heterozygous knockout mice to irradiation-induced alveolitis and are corroborated by the increased radiosensitivity in vitro of (-/-) and (-/+) human MnSOD transgene. In the second specific aim we will determine the level of over- expression of MnSOD which correlates with the relative lung radioresistance in FeVB/NHsd transgenic mice over-expressing the human MnSOD transgene in the airway linked to the lung parenchymal cell-specific